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REVIEW

Abiotic conditions governing the myceliogenic germination of *Sclerotinia sclerotiorum* allowing the basal infection of *Brassica napus*

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Abstract
Sclerotinia sclerotiorum is the causal pathogen of sclerotinia stem rot (SSR) in canola, causing significant yield losses in this crop globally, when conditions are favourable. The pathogen can cause disease symptoms on canola through ascospore infection from carpogenically germinated sclerotia or by basal infection from myceliogenically germinated sclerotia. While infection through carpogenic germination in canola is the main mode of infection and well-studied, little is known about myceliogenic germination of sclerotia and subsequent basal infection of canola. This review describes the lifecycle of *S. sclerotiorum* on canola and presents an overview of the current knowledge of the factors that influence myceliogenic germination. These include factors such as sclerotium maturity, rind melanisation, temperature and moisture or a combination of these factors. Subsequently, the most likely avenues of *S. sclerotiorum*-based basal infection in canola are discussed and compared to basal infection in other host crops. We conclude that myceliogenic germination can be promoted by incubation of sclerotia at extreme temperatures followed by exposure to moisture. Future research to determine the prevalence of myceliogenic germination and subsequent basal infection of canola in the field is required.

Keywords *Sclerotinia sclerotiorum* · Canola · Myceliogenic germination · Carpogenic germination · Basal infection · Sclerotinia stem rot

Introduction

The plant pathogen *Sclerotinia sclerotiorum* (Lib.) de Bary is a cosmopolitan broad host range pathogen infecting over 400 plant species, which include agricultural broad-leafed crops such as sunflowers, carrots, canola and various pulses including chickpea, faba beans, field peas, lentils, lupins and peanuts (Boland and Hall 1994). This wide host range makes it a difficult disease to control through crop rotation, especially since its resting structures (termed sclerotia) can survive in the soil for several years. *S. sclerotiorum* is one of the major pathogens of canola (*Brassica napus* L.), also known as rape or oilseed rape, which is a member of the cabbage family, the Brassicaceae. Oilseed rape is primarily produced in the form of canola (Derbyshire and Denton-Giles 2016), because it has low concentrations of erucic acid and glucosinolates (double low), which are cytotoxic (Gunstone 2001). Canola is a globally grown crop for the production of oil and meal, with uses in the production of edible vegetable oil, animal feed, aquaculture and biodiesel. It is the second largest meal crop behind soybean and can be grown as a winter or a spring crop. Major producers globally are Australia, Canada, China, the European Union and India with Canada producing 18.4 million tons in 2016 alone (FAOSTAT 2018). Considering canola's commercial status, it is advantageous to continue research into increasing crop yield. Disease, in particular sclerotinia stem rot (SSR) is a major detriment to canola yield (Sharma et al. 2016; Twengström et al. 1998; Wang et al. 2009). This disease is caused by the fungus *S. sclerotiorum*, which is prevalent in all main canola production areas (Derbyshire and Denton-Giles 2016). It was estimated that SSR causes a financial loss of approximately $AUD 10.1

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million and US$ 8.5 million annually in Australia and North Dakota respectively (del Río et al. 2007; Murray and Brennan 2012). While no major genetic resistance has been identified as a means to control the disease in commercial canola varieties (Rana et al. 2017; Uloth et al. 2015), evidence is emerging of partial resistance in *Brassica* germplasm (Denton-Giles et al. 2018; Mei et al. 2011; Rana et al. 2017; Uloth et al. 2013, 2015;). Partial resistance to *S. sclerotiorum* is controlled by various quantitative trait loci (QTLs) (Behla et al. 2017; Gyawali et al. 2016; Li et al. 2015; Wei et al. 2014; Zhao and Meng 2003; Zhao et al. 2006) and could offer a means to reduce the impact of SSR in canola. The pathogen can infect canola in two different ways, either through ascospores that arise as a result of carpogenic germination of sclerotia or basal infection from myceliogenically germinated sclerotia. While infection of canola most commonly occurs through carpogenic germination, the role of myceliogenic germination and basal infection in canola is poorly understood. This review will describe the lifecycle of *S. sclerotiorum* in canola through both infection methods, followed by a comprehensive summary of the abiotic conditions governing the myceliogenic germination of *S. sclerotiorum* sclerotia. Subsequently, we describe the possible infection pathway and conclude by comparing and contrasting basal infection strategies of *S. sclerotiorum* in other crops to that of canola and propose future strategies to further characterize basal infection by *S. sclerotiorum* in canola.

The two lifecycles of *Sclerotinia sclerotiorum*

The lifecycle of *S. sclerotiorum* (Fig. 1), depends on the mode of infection and is therefore related to the type of sclerotium germination. *S. sclerotiorum* survives in the soil as hard black dormant fungal structures termed sclerotia, which can germinate carpogenically (Derbyshire and Denton-Giles 2016; Sharma et al. 2016). Carpogenic germination is characterised by the release of thousands of airborne spores (ascospores) from small mushroom-like structures called apothecia (Sharma et al. 2016). The wind-borne ascospores infect the petals which subsequently fall into the lower crop canopy. The flowering period is critical for SSR to occur, since the senescent petals support the growth of mycelium for subsequent infection of the canola stem (Purdy 1979). SSR is characterised by brown lesions on the stem, pods and branches of *B. napus*, which turn a grey colour over time. Extensive infection causes canola to wilt and ripen prematurely (Khangura and Beard 2015). Sclerotia form within the stem as SSR progresses and can also be visible outside the stem in highly humid conditions. These sclerotia end up in the soil through plant death and stem lodging or during harvest, completing the lifecycle. Lodging of the stem occurs when necrotic lesions become established and cause the stem to lose rigidity (Derbyshire and Denton-Giles 2016). SSR is primarily controlled by the prophylactic application of fungicides, where timing of the application is key to successful control of SSR (Bradley et al. 2006; Derbyshire and Denton-Giles 2016). This is because SSR outbreaks are difficult to predict, especially since initial infection of the petals is asymptomatic, thus making the optimal time for spraying a challenging decision. SSR of canola initiated by carpogenic germination of *S. sclerotiorum* has been well documented since it is thought to be the main cause of infection in canola fields (Garg et al. 2010; Huang and Dueck 1980). However, *S. sclerotiorum* sclerotia are capable of another type of germination, resulting in an alternative infection pathway (Bardin and Huang 2001).

Unlike the infection of above-ground tissue via carpogenic germination, *S. sclerotiorum* can infect canola directly from the ground by producing mycelia that can penetrate host tissues, through a process termed myceliogenic germination (Finlayson et al. 1989; Purdy 1979). Myceliogenic germination is more commonly associated with the small sclerotia of *Sclerotinia minor*, but has also been reported for *S. sclerotiorum* (Purdy 1979). Infection via myceliogenic germination is termed basal infection and results in SSR. Basal infection of canola is known to occur at ground-level or beneath the soil surface for other crops (Sharma et al. 2016). While the appearance of lesions arising from basal infection may differ between crops, it can be identified by the patchy production of fluffy white mycelia at the base of the stem (Bolton et al. 2006). Fungicide application has been established to control SSR occurring via carpogenic germination, while the efficacy of fungicide use has not been determined for SSR caused by basal infection from myceliogenic germination. In a conference proceeding by Khangura (2017), it was reported that the number of locations in Western Australia (WA) with basal infection from *S. sclerotiorum* had dramatically increased from 2015 to 2016. There is minimal literature addressing basal infection of canola, possibly because direct infection from myceliogenic germination is not considered the common cause of SSR (Khangura and Beard 2015). While basal infection of canola is not the main mode of infection by SSR it has been hypothesized to be more common in crops used in rotation with canola, such as chickpeas and lupins in Australia (Khangura and Beard 2015). Since multiple crops in a rotation are hosts for *S. sclerotiorum*, one could speculate this could lead to the number of soil-borne sclerotia increasing in a paddock, leading to a higher frequency of basal infection in canola. While this requires further investigation, there is some literature on myceliogenic germination, which is the essential precursor to basal infection which will be discussed in detail below.

Factors that can promote myceliogenic germination

The type of sclerotial germination (e.g. carpogenic or myceliogenic germination) depends on the environmental conditions (Abawi and Grogan 1979). For ascospore infection from carpogenically germinated sclerotia, long humid and wet conditions are favourable, resulting in outbreaks of SSR (Hind-Lanoiselet and Lewington 2004). Suitable environmental conditions are also responsible for basal infection often seen as the abundant growth of white mycelia on the canola stem (Purdy 1979). While these general environmental conditions that lead to the onset of disease symptoms have been described, multiple studies have also endeavoured to determine which specific abiotic conditions support myceliogenic germination of *S. sclerotiorum*, most of which were conducted in vitro. A summary of these studies will be described in the subsequent paragraphs.

The role of sclerotium maturity and rind melanisation on myceliogenic germination

Sclerotium maturity is a key factor influencing the ability of myceliogenic germination to occur. Immature sclerotia are less pigmented than mature sclerotia, due to the deposition of melanin into the rind during formation (Huang and Kozub 1994; Jones 1970). Before pigmentation begins, sclerotia initials can be identified by the formation of small liquid sacs on dense mats of white mycelia (Huang and Kozub 1994). These liquid sacs (termed exudate) disappear 96–144 h after the initials form and arise from internal hydrostatic pressure (Al-Hamdani and Cooke 1987; Huang and Kozub 1994). Immature sclerotia myceliogenically germinate more readily than fully melanised mature sclerotia (Huang and Kozub 1994). This occurs because the melanin rind controls sclerotial dormancy and acts as a physical barrier to myceliogenic germination (Coley-Smith and Cooke 1971; Huang 1985). Huang (1985) demonstrated that the frequency of myceliogenic germination could be increased on moist sand by artificially injuring a fully melanised rind or by using sclerotia with less rind pigmentation. However, infection of host plants from myceliogenically germinated immature sclerotia in the field would be rare, since the reduced rind makes them more vunerable to invasion by microorgansims. Furthermore, wet and humid conditions can initiate re-melanisation of the rind, making the sclerotia dormant again (Huang 1985). In summary, the melanised rind layer of a sclerotium acts to inhibit myceliogenic germination.

The role of temperature on myceliogenic germination

A fully melanised sclerotial rind is important for controlling dormancy and temperature has been shown to alter the deposition of melanin into the rind cells (Huang and Kokko 1989). Huang and Kokko (1989) determined that sclerotia formed at low-temperatures (7 °C) undergo delayed melanisation, resulting in sclerotia with unmelanised or partially melanised rind cells. Yet, all sclerotia could myceliogenically germinate on moist sand, regardless of the formation temperature (7, 16 and 30 °C) and a sufficient explanation for the similar myceliogenic germination frequencies observed was not reported. In a subsequent study, Huang (1991) tested the effect of cold conditioning on the myceliogenic germination of *S. sclerotiorum*. Sclerotia stored at sub-freezing temperatures (−10 and − 20 °C) were all able to germinate myceliogenically (on moist sand), with sclerotia stored at −20 °C germinating more readily. It was suggested that the prolonged freezing may cause injury to the sclerotium rind promoting myceliogenic germination, however no evidence was provided to support this hypothesis. It was suggested that the conditioning of sclerotia at sub-freezing temperatures could induce a change from carpogenic to myceliogenic germination. In contrast, a study by Foley et al. (2016) demonstrated that high frequencies of myceliogenic germination were not induced by conditioning sclerotia at −20 °C; regardless of whether sclerotia were dry or hydrated.

While sub-freezing temperature conditioning could occur in canola cropping regions in the northern parts of the Americas, it does not represent the conditions observed in the canola growing regions of Australia. In Western Australia for example, sub-freezing temperatures are rare, though high temperature extremes are common. The effects of high temperatures on myceliogenic germination of sclerotia were recently investigated by Lane et al. (2018). They demonstrated that conditioning of sclerotia through both sub-freezing (−20 °C) and heat drying (37 °C) led to consistent myceliogenic germination (Lane et al. 2018). In conclusion conditioning of sclerotia with extreme temperatures (e.g − 20 °C and 37 °C) can lead to consistent levels of myceliogenic germination. Whether these temperature extremes lead to sclerotial injury that initiate myceliogenic germination warrants further research.

The role of both temperature and moisture on myceliogenic germination

Some studies have investigated how combinations of temperature and soil moisture influence the ability of *S. sclerotiorum* to germinate myceliogenically. For instance, Hao et al. (2003) found that myceliogenic germination of sclerotia in Petri dishes with two different soil types was infrequent irrespective of the soil moisture (0 to −0.3 MPa) and temperature (10–30 °C) combination applied. Matheron and Porchas (2005) conducted a similar study, measuring myceliogenic germination frequency under different soil moisture and temperature

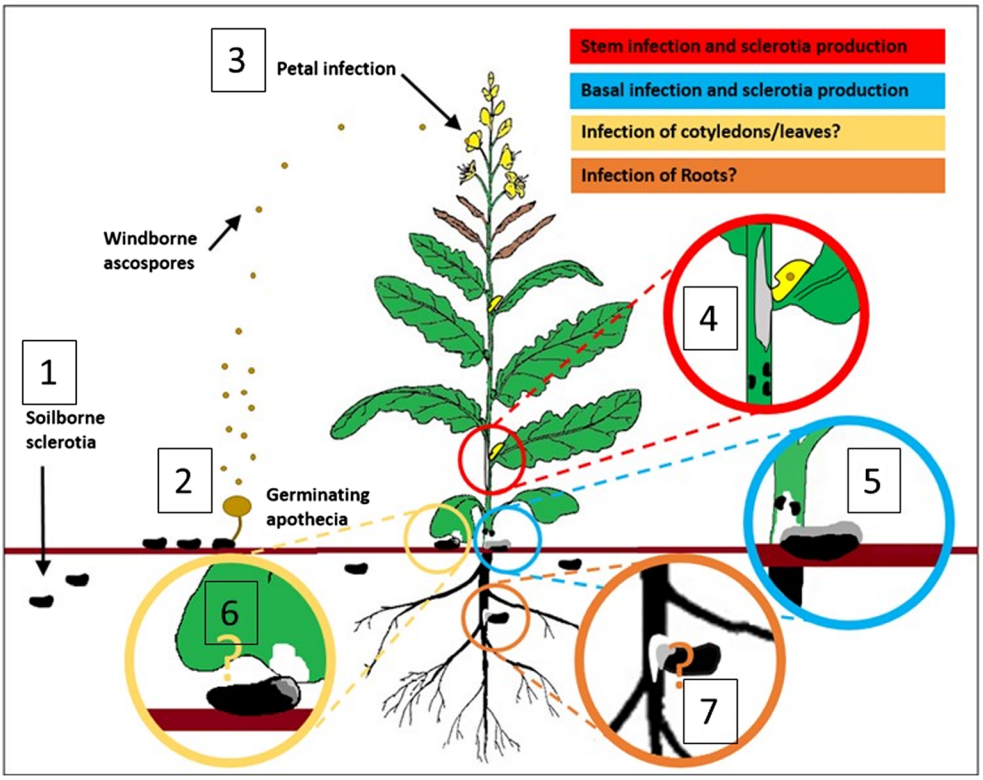

Fig. 1 The *S. sclerotiorum* life cycle, displaying the main carpogenic infection pathway and and potential basal infection pathways from myceliogenically germinated sclerotia in canola. Sclerotia can survive in the soil for several years and can germinate carpogenically when conditions become favourable (1). Carpogenic germination results in the formation of apothecia which harbour the ascospores (2). The ascospores are wind dispersed onto the flower petals (3), where infected petals fall into leaf axials, allowing the contact of the mycelial inoculum with the stem (4). Alternatively, the sclerotia may germinate myceliogenically, infecting the stem directly from the soil-line, this is termed basal infection (5). Secondary sclerotia are also formed from this infection method. Possible infection pathways similar to basal infection include direct infection of a canola leaf from a sclerotium resting on the soil (6) and direct infection of the canola roots from a sclerotium below the soil surface (7). The image is adapted from Derbyshire and Denton-Giles (2016)

treatments. However, after these treatments, sclerotia were sterilised and placed on acidified Potato Dextrose Agar (PDA). Therefore, Matheron and Porchas (2005) actually measured sclerotium viability, since all viable sclerotia produce mycelia on PDA. This is because PDA is a rich nutrient source promoting hyphal growth from sclerotia. This limitation was noted by Matheron and Porchas (2005), stating that they were only able to determine the proportion of sclerotia capable of myceliogenic germination under artificial conditions. Nonetheless, the results reveal that sclerotium viability tended to decrease in wet soil as temperature increased, with no growth of sclerotia on acidified PDA after two weeks in wet soil, at 40 °C (Matheron and Porchas 2005). These results are similar to the findings by Hao et al. (2003), demonstrating that the viability of sclerotia decreased with increased temperature, for specific soil moisture levels. Clearly, the interaction of soil moisture and temperature on sclerotium viability needs to be reassessed, with a thorough report on the myceliogenic germination frequency, occurring naturally in the soil.

In an alternative study by Huang et al. (1998) the myceliogenic germination of sclerotia with variable water contents was tested. It was revealed that high formation temperatures (20 and 25 °C), used in combination with sclerotial desiccation, caused sclerotia to readily myceliogenically germinate on moist sand in high humidity. Non-desiccated sclerotia grown on PDA can germinate in 100% relative humidity on moist sand. However, they only produce short sparse hyphae compared with the vigorous myceliogenic germination of desiccated sclerotia. Similar results were not produced by Foley et al. (2016) in their re-evaluation, but it was suggested that prolonged desiccation may have facilitated vigorous myceliogenic germination that was demonstrated by Huang et al. (1998). It was proposed that the drying process caused injury to the rind, which subsequently allowed the leakage of nutrients, supporting vigorous hyphal growth (Huang et al. 1998). This theory is supported by the previous finding that dried sclerotia when remoistened, release nutrients, however this is also associated with sclerotium degradation due to colonisation by microorganisms (Smith 1972). Consequently, sclerotium desiccation may be a consistent method for inducing myceliogenic germination compared to soil moisture treatments, however its relevance to the field is undermined by the decreased survival during re-moistening.

The role of moisture on myceliogenic germination

Moisture is a key factor influencing the survival of *S. sclerotiorum* sclerotia. In flooded soils sclerotia have a lower survival rate compared to dry soil (Coley-Smith and Cooke 1971). Although sclerotia are degraded in high moisture content soils, this is offset by the production of secondary sclerotia, where a balance of destruction and formation is achieved in completely saturated soil (Williams and Western 1965). Al-Hamdani and Cooke (1987) found that decreasing the water potential of a nutritional medium decreases the radial growth of *S. sclerotiorum*. Additionally, when water potential decreased, the sclerotium size increased, since more nutrients were allocated to the secondary sclerotia and there were fewer sclerotia produced. Al-Hamdani and Cooke (1987) also evaluated the myceliogenic germination of desiccated *S. sclerotiorum* sclerotia on 2% water agar; where the density of mycelia produced increased with decreasing original sclerotium formation water potential. Variations in soil moisture are often used in experiments to replicate field conditions rather than directly changing sclerotial water content, using free-water or drying (Ferraz et al. 1999; Hao et al. 2003; Matheron and Porchas 2005; Nepal and del Río Mendoza 2012). Nepal and del Rio Mendoza (2012) showed that dry sclerotia become fully saturated within 25 h (depending on sclerotium size), regardless of soil saturation (minimum 25%). However, both the carpogenic germination and viability of sclerotia were affected by variable soil moisture contents (Hao et al. 2003; Matheron and Porchas 2005), indicating that the surrounding soil moisture is of greater importance to germination and survival of a sclerotium than the water content of the individual sclerotium. This theory is emphasised by the fact that sclerotia kept in flooded soil (at variable temperature) for three weeks by Matheron and Porchas (2005), were not viable and was associated with sclerotial disintegration. Saturation of sclerotia through imbibing is known to increase carpogenic germination (Nepal and del Río Mendoza 2012), however its effect on myceliogenic germination has not been well studied, whereas the effect of desiccated, dried and fresh sclerotia have been investigated (Foley et al. 2016; Huang et al. 1998). In a recent study by Lane et al. (2018) it was demonstrated that submerging sclerotia in water with exposure to either freezing temperatures or heat stress led to consistent myceliogenic germination. In summary, water availability has significant influence over the biological processes of *S. sclerotiorum*, whether it be melanisation, production of exudate and secondary sclerotia or density of mycelia during myceliogenic germination.

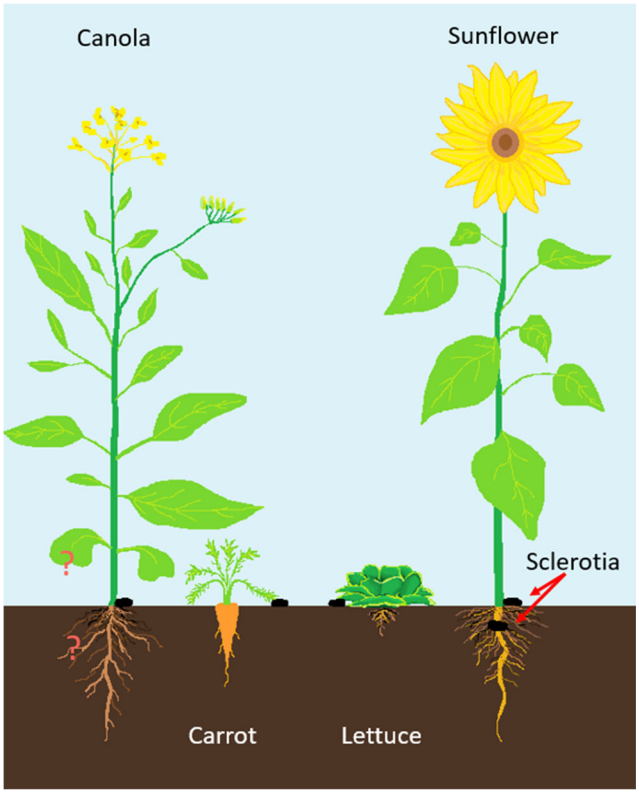

Fig. 2 Common infection pathways for different crops, visualised by the *S. sclerotiorum* sclerotium locations, which are known to cause infection from myceliogenic germination. In canola, basal infection occurs when sclerotia myceliogenically germinate at the base of the stem, but it remains unclear if basal infection can occur through the leaves and roots (indicated by the questions marks). In carrot, the principal infection pathway is through the leaves, when they rest on the soil in proximity to a sclerotium. For lettuce, infection also occurs primarily through the leaves. Infection in sunflower occurs when a sclerotium either infects the roots or the hypocotyl and stem base. The question marks denote other canola organs that might facilitate basal infection (through the roots and leaves)

Basal infection of canola by *Sclerotinia sclerotiorum*

Very few papers have examined the ability for myceliogenically germinating *S. sclerotiorum* sclerotia to infect canola. Those that have, didn't provide a comprehensive analysis of the possible infection pathways. Li et al. (2008) observed basal infection of *B. napus,* from myceliogenically germinating sclerotia in a field study. Although it was assumed that infection occurred at the stem's base, a controlled inoculation assay was not carried out. Huang and Dueck (1980) demonstrated that basal infection of *B. napus* can occur at low frequencies, by using whole sclerotia placed at the stem's base (Figs. 1 and 2). Furthermore, Lane et al. (2018) demonstrated that myceliogenically germinated sclerotia are able to infect healthy mature leafs under controlled conditions. However, infection through alternative plant organs, including senescing leaves, hypocotyls and roots (Fig. 1) was not studied. Further research needs to be conducted to demonstrate the optimal infection pathway enabling basal infection.

Basal infection pathways in other crops by *Sclerotinia sclerotiorum*

In other crop plants, basal infection is a major contributor to disease caused by *S. sclerotiorum* as schematically presented in Fig. 2. For example, Finlayson et al. (1989) showed that *S. sclerotiorum* primarily causes infection in carrots when mycelium contacts the leaves. The mycelium can then move through the plant organs and enter the roots, spreading the infection. In sunflower, myceliogenically germinating sclerotia can directly infect the roots (Huang and Hoes 1980). However, Finlayson et al. (1989) discovered that the same did not apply to carrots, subsequently suggesting that the sunflower root exudate is responsible for stimulating myceliogenic germination. *S. sclerotiorum* can myceliogenically germinate in the soil and infect the hypocotyl and stem of sunflower seedlings directly without prior wounding; this produces the characteristic wilt symptom (Huang and Dueck 1980). Mycelial infection from *S. sclerotiorum* is uncommon in lettuce compared with infection from ascospores (Patterson and Grogan 1985). However, mycelial infection can occur through the leaves, which is followed by wilting and watery decay (Subbarao 1998). Dense white mycelium forms on the infected tissues and secondary sclerotia form on the leaves touching the soil, around the crown, and in the tap-root (Subbarao 1998). Together these studies suggest that the optimal basal infection pathway is dependent on the host crop (Fig. 2).

Conclusion

Research into the abiotic conditions promoting myceliogenic germination is essential for controlling the basal infection of canola by *S. sclerotiorum*, because changes in environmental conditions play a key role in breaking sclerotial dormancy (Garg et al. 2010; Huang 1985). Rind injury (Huang 1985), exposure to extreme temperatures (Huang 1991; Lane et al. 2018) and the availability of moisture (Al-Hamdani and Cooke 1987; Lane et al. 2018) are all important factors able to influence myceliogenic germination. Myceliogenic germination and subsequent basal infection are relevant to all canola growing regions of the world. While *S. sclerotiorum* infection pathways observed in other crops may provide insight into how the phytopathogen preferentially infects canola through basal infection, further research needs to be initiated to understand how this commonly occurs in this agriculturally important crop. Determining the basal infection pathways and yield loss as a consequence of this mode of infection is likely to lead to novel integrated disease management methods for the control of this disease.

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